cachexia group(374.44+13.57 ng/ml) was lower than in the heart failure non cachexia group (421.80+9.14 ng/ml) (P <0.05), (5) the expression of p85, p-Akt and NF-κB: in cardiac cachexia group was lower than in the heart failure non cachexia group (P <0.05) and control group (P <0.05), but the heart failure non cachexia group was higher than the control group (P <0.05); (6) the expression of caspase9 mRNA: in cardiac cachexia group(1.38+0.03) was higher than in the heart failure non cachexia group(0.49+0.04) and control group(0.62+0.07) (P<0.05), (7) the expression of Bcl-xl mRNA: in cardiac cachexia group (1.39+0.20) was lower than in the heart failure non cachexia group(8.03+0.28) and control group(7.80+0.15) (P<0.05).

CONCLUSIONS (1) There were insulin and GH resistance in the heart failure cachexia patients, (2) PI3K/Akt signal transduction pathways was activated by phosphorylation in heart failure, which is play a protective effect on the heart when combined with cachexia. and the expression of p85, p-Akt and NF-κB were decreased, illustrate that PI3K/Akt signal pathway was restrained, which eventually lead to cell apoptosis. (3) PI3K/Akt signal transduction pathway may be a new therapeutic target in cardiac cachexia.

GW26-e0249

Effect of Eplerenone on Plasma TGF-β1 level in Patients with Chronic Heart Failure

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OBJECTIVES Renin - angiotensin - aldosterone system (RAAS), by affecting the nervous hormone levels, involves in the development of chronic heart failure(CHF). Of interest, for patients with moderate to severe chronic heart failure, on top of angiotensin-converting enzyme inhibitors (ACEI) and beta blockers, eplerenone administration will further reduce the total mortality. However, the therapeutic mechanism for CHF with eplerenone administration is not yet fully understood.

METHODS Renin - angiotensin - aldosterone system (RAAS), by affecting the nervous hormone levels, involves in the development of chronic heart failure(CHF). Of interest, for patients with moderate to severe chronic heart failure, on top of angiotensin-converting enzyme inhibitors (ACEI) and beta blockers, eplerenone administration will further reduce the total mortality. However, the therapeutic mechanism for CHF with eplerenone administration is not yet fully understood.

RESULTS Compared with conventional treatment, LVEF and 6-minute walk test(6-MWT) distances increased, while LVP, LVEDD, IVS, TGF-β_1 level, BNP level and the blood pressure all reduced in patients received eplerenone administration (P<0.05), TGF-β_1 levels were inversely correlated with LVEF, and positively correlated with BNP level (P<0.05).

CONCLUSIONS Eplerenone can reduce plasma TGF-β1 level by reversing cardiac remodeling and improve cardiac function in chronic heart failure patients.

GW26-e4402

Silencing microRNA-155 reduces LPS-induced cardiac apoptosis via targeting pea15a

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OBJECTIVES Sepsis-related cardiac dysfunction is characterized by inflammation and metabolic repression. microRNAs, a small non-coding RNA, inhibit mRNA translation or promote mRNA degradation through pairing to the 3'-UTR of target genes mRNA. MiR155 is up-regulation as a continual feature of the mammalian inflammatory response. In this study, we tested the hypothesis that miR155 regulates in heart dysfunction with sepsis.

METHODS E.coli lipopolysaccharide (LPS)(5mg/kg) was administered to C57BL/6 mice to induce a sepsis-induced cardiac dysfunction model within 5-7 h. Cardiac function was assessed by Echocardiography 5~6h post-LPS administration. Myocardium were obtain within 7~9h after LPS treatment for gene expression and protein analysis. A systematic analysis of cardiac miRNA profiles using an established miRNAarray was performed to assess dys-regulated miRNAs in sepsis-induced cardiac dysfunction. To forced expression of miR155, miR155 agomirs were injected in the tail vein of C57B1/6J mice on 3 consecutive days with a total of 30 mg/kg agomir and inhibition of miR155 in vivo by 80 mg/kg 155 antagomir followed by LPS administration.

RESULTS LPS induced reduction 15% in Fractional shortening (%FS) and 25% in ejection fraction (%EF). Expression of miR155 was increased by 2 fold. Over-expressing of miR155 with systemic delivery of agomir led to 5% decrease in FS and 10% in EF as compared to scramble control. Aggravation of the LPS induced cardiac dysfunction by miR155 agomir is not associated with alteration in inflammation or cardiac metabolism. MiR155 agomir increased LPS- induced myocardium apoptosis and increased ratio of Bax/Bcl-2 at the protein level. Deficiency of miR155 markedly rescued the LPS induced heart failure and apoptosis. In vivo, western bloting found that over-expression of miR155 led to significantly inhibition of Pea15a in mice. Using bioinformatics analyses and validated luciferase reporter assays, Pea15a was identified as a novel miR155-target. Finally, we observed that critically ill patients with sepsis had increased levels of miR155 compare with healthy control as well.

CONCLUSIONS The present study demonstrated that miR155 regulates sepsis-related cardiac apoptosis by target gene Pea15a. Finally, our results identify inhibition of miR-34a as a potential therapeutic strategy to improve sepsis -induced heart failure.

GW26-e4801

The correlations between iron metabolism and myocardial energy expenditure in patients with chronic heart failure

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OBJECTIVES Chronic heart failure (CHF) is a major public health burden worldwide and is associated with high morbidity, mortality and cost. Recent study demonstrated that iron metabolism and myocardial energy expenditure (MEE) were altered in CHF patients. In this study, we aimed to analyze the effects, clinical significance, and possible correlations of iron metabolism on MEE in patients with CHF.

METHODS We recruited 96 CHF patients [age: 67.4±11.5 years, males: 61.5%, New York Heart Association (NYHA) class (II/III/IV): 45/36/15] from the Cardiology Department in Nanfang Hospital, Southern Medical University from January 2014 to January 2015. The concentrations of serum hemoglobin, Fe, total iron-binding capacity, transferrin saturation, transferrin, soluble transferrin receptor, ferritin, and Pro-BNP were evaluated. Echocardiography was used to assess LA, LV, PWTs, LVIDs, LVIDd, LVEF, LVFS, and MEE. Iron deficiency was defined as ferritin <100 ng/mL or 100-300 ng/mL with transferrin saturation <20%.

RESULTS The patients were divided into iron-deficient and iron-sufficient groups; the incidence of iron deficiency in all subjects was 38.5%. The differences of demographic characteristics (age, sex and BMI) and HGB concentrations in two groups were similar (P > 0.05). Interestingly, MEE was significantly higher in the iron-deficient group [64.963 (51.555 to 78.300) cal/min vs. 40.176 (25.346 to 56.914) cal/min, P < 0.001]. Similarly, MEE in patients with NYHA classes II and III was significantly higher in the iron-deficient group (55.6±11.4 cal/min vs. 39.7±17.5 cal/min, P=0.002; 63.6±16.9 cal/min vs. 42.6±21.0 cal/min, P=0.003). Bivariate analysis confirmed that MEE was significantly correlated with ferritin (r=-0.406, P < 0.001), transferrin saturation (r=-0.307, P=0.002), Pro-BNP (r=0.333, P=0.001), NYHA class (r=0.455, P < 0.001), LVEF (r=-0.477, P < 0.001), LVFS (r=-0.657, P < 0.001), LV (r=0.770, P < 0.001), and LVIDd (r=0.748, P < 0.001). Multiple linear regression analysis on the above variables showed that iron deficiency, higher LVIDd, NYHA class, and lower LVFS predicted higher MEE (r^2=0.748, P < 0.001).

CONCLUSIONS Iron deficiency may play an important role in the disorders of MEE in CHF patients, regardless of baseline HGB concentrations.

CONGENITAL HEART DISEASE AND INTERVENTIONS

GW26-e0786

Prediction of Spontaneous closure in isolated ventricular septal defects by fetal echocardiography

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OBJECTIVES By conducting a follow-up survey in children who were diagnosed with simple ventricular septal defect(VSD) in their mothers' second trimester through fetal echocardiography, we hope